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Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization.
J Appl Physiol. 1977 Oct;43(4):700-3.
PMID: 908686 [PubMed - indexed for MEDLINE]

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☐ 1: J Appl Physiol. 1977 Oct;43(4):700-3.

Related Articles, Links

Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization.

MacDougall JD, Ward GR, Sale DG, Sutton JR.

Nine healthy subjects were studied under control conditions and following 5 mo of heavy resistance training and 5 wk of immobilization in elbow casts. Needle biopsies were taken from triceps brachii and analyzed for adenosine triphosphate (ATP), adenosine diphosphate (ADP), creatine (C), creatine phosphate (CP, and glycogen concentrations. Training resulted in an 11% increase in arm circumference and a 28% increase in maximal elbow extension strength. Immobilization resulted in decreases in arm circumference and elbow extension strength of 5% and 35%, respectively. Training also resulted in significant increases in resting concentrations of muscle creatine (by 39%), CP (by 22%), ATP (by 18%), and glycogen (by 66%). Conversely, immobilization significantly reduced CP concentration by 25% and glycogen concentration by 40%. It was concluded that heavy-resistance training results in increases in muscle energy reserves which may be reversed by a period of immobilization-induced disuse.

PMID: 908686 [PubMed - indexed for MEDLINE]

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Muscle unloading induces slow to fast transitions in myofibrillar but not mitochondrial properties. Relevance to skeletal muscle abnormalities in heart failure.

Bigard AX, Boehm E, Veksler V, Mateo P, Anflous K, Ventura-Clapier R.

Unite de Bioenergetique, CRSSA, La Tronche, France.

Muscle deconditioning is a common observation in patients with congestive heart failure (CHF), chronic obstructive pulmonary disease, neuromuscular diseases or prolonged bed rest. To gain further insight into metabolic and mechanical properties of deconditioned slow-twitch (soleus) or fast-twitch (EDL) skeletal muscles, we induced experimental muscle deconditioning by hindlimb suspension (HS) in rats for 3 weeks. Cardiac muscle was also studied. Besides profound muscle atrophy, increased proportion of fast type II fibers as well as fast myosin isoenzymes, we found decreased calcium sensitivity of Triton X-100 skinned fiber bundles of soleus muscle directed towards the fast muscle phenotype. Glycolytic enzymes such as hexokinase and pyruvate kinase were increased, and the LDH isoenzyme pattern was clearly shifted from an oxidative to an anaerobic profile. Creatine kinase (CK) and myokinase activities were increased in HS soleus towards EDL values. Moreover, the M-CK mRNA level was greatly increased in soleus, with no change in EDL. However, oxygen consumption rate assessed in situ in saponin skinned fibers (12.5 +/- 0.8 in C and 15.1 +/- 0.9 micromol O2/min/g dw in HS soleus compared to 7.3 +/- 1.3 micromol O2/min/g dw in control EDL), as well as mitochondrial CK (mi-CK) and citrate synthase activities, were preserved in HS soleus. Following deconditioning no change in Km for ADP of mitochondrial respiration, either in the absence (511 +/- 92 in C and 511 +/- 111 microM in HS soleus compared to 9 +/- 4 microM in control EDL) or presence of creatine (88 +/- 10 in C and 95 +/- 16 microM in HS soleus compared to 32 +/- 9 microM in control EDL), was found. The results show that muscle deconditioning induces a biochemical and functional slow to fast phenotype transition in myofibrillar and cytosolic compartments of postural muscle, but not in the mitochondrial

compartment, suggesting that these compartments are differently regulated under conditions of decreased activity.

PMID: 9925374 [PubMed - indexed for MEDLINE]

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Potential benefits of creatine monohydrate supplementation in the elderly.

Tarnopolsky MA.

Dept of Neurology/Neurological Rehabilitation, McMaster University Medical Center, Hamilton, Ontario, Canada. tarnopol@fhs.mcmaster.ca

Creatine plays a role in cellular energy metabolism and potentially has a role in protein metabolism. Creatine monohydrate supplementation has been shown to result in an increase in skeletal muscle total and phosphocreatine concentration, increase fat-free mass, and enhance high-intensity exercise performance in young healthy men and women. Recent evidence has also demonstrated a neuroprotective effect of creatine monohydrate supplementation in animal models of Parkinson's disease, Alzheimer's disease, amyotrophic lateral sclerosis, and after ischemia. A low total and phosphocreatine concentration has been reported in human skeletal muscle from aged individuals and those with neuromuscular disorders. A few studies of creatine monohydrate supplementation in the elderly have not shown convincing evidence of a beneficial effect with respect to muscle mass and/or function. Future studies will be required to address the potential for creatine monohydrate supplementation to attenuate age-related muscle atrophy and strength loss, as well as to protect against age-dependent neurodegenerative disorders such as Parkinson's disease and Alzheimer's disease.

Publication Types:
- Review
- Review, Tutorial

PMID: 11085837 [PubMed - indexed for MEDLINE]

1: J Rheumatol. 1996 Apr;23(4):603-8.

Comment in:

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Low serum creatine kinase activity is associated with muscle weakness in patients with rheumatoid arthritis.

Stucki G, Bruhlmann P, Stoll T, Stucki S, Willer B, Michel BA.

Department of Rheumatology and Physical Medicine, University Hospital Zurich, Switzerland.

OBJECTIVE. In rheumatoid arthritis (RA) serum creatine kinase (CK) is reduced in association with inflammatory response variables. Our objective was to examine whether low CK is associated with muscle weakness and to what extent the hypothesized relationship between CK and muscle weakness can be explained by anthropometric and sociodemographic variables and/or disease variables. METHODS. Cross sectional and longitudinal retrospective analyses of clinical, radiological, and biochemical data of a prospective cohort of consecutive patients with RA. Isometric muscle strength was measured with a validated muscle strength index (MSI); CK was measured with an enzymatic assay (N-acetyl-cysteine, 37 degrees C). RESULTS. 65 patients were enrolled in the study and we obtained complete one year followup data from 47. In cross sectional analysis, CK was a significant, moderate correlate of the MSI ($r = 0.43$, $p < 0.01$). CK remained a significant explanatory variable of the MSI in multivariate models that controlled for demographic variables and lean body mass, corticosteroid use, and biochemical, clinical, and radiological disease variables. In longitudinal dichotomous analyses, worsening in CK was weakly but significantly associated with decreased muscle strength, whereas in linear analyses the association did not reach significance. CONCLUSION. In patients with RA, low CK activity is associated with muscle weakness. Demographic, anthropometric, and disease variables related to muscle mass or muscle atrophy explain only part of this association. Our findings support the hypothesis that muscle weakness may be partly caused by a disease related reduction of CK activity independent of muscle atrophy.

PMID: 8730112 [PubMed - indexed for MEDLINE]

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